AMENDMENTS TO THE CLAIMS

1. (Currently amended) A method for preventing or treating diabetes in a mammal, the method comprising administering to the mammal a therapeutically effective amount of at least one GLP-1 agonist ~~or a related molecule having GLP-1 effect~~, wherein the amount and timing of administration are such as to prevent or treat diabetes or related disorder in the mammal without the continuous presence of the agonist ~~molecule~~.

2. (Currently amended) The method of claim 1, wherein the method further comprises reducing administration of the GLP-1 agonist ~~or related molecule~~ below about the therapeutically effective amount for a time conducive to producing a drug holiday, the method being sufficient to prevent or treat the diabetes or related disorder in the mammal.

3. (Currently amended) The method of claim 2, wherein administration of the GLP-1 agonist ~~or related molecule~~ is reduced during the drug holiday by at least about 50% below the therapeutic amount.

4. (Currently amended) The method of claim 3, wherein administration of the GLP-1 agonist ~~or related molecule~~ is reduced during the drug holiday by at least about 90% below the therapeutic amount.

5. (Currently amended) The method of claim 4, wherein administration of the GLP-1 agonist ~~or related molecule~~ is stopped during the drug holiday.

6. (Currently amended) The method of claims 1-5, wherein during the drug holiday is further defined as a time interval between a first endpoint following the reduction in administering the GLP-1 agonist ~~or related molecule~~ and a second endpoint.

7. (Original) The method of claim 6, wherein the second endpoint is identified by a standard FBG or glycosylated hemoglobin test.

8. (Previously presented) The method of claim 1, wherein the drug holiday is for about one day to about twenty five weeks.

9. (Original) The method of claim 8, wherein the drug holiday is for between from about three to four weeks.

10. (Currently amended) The method of claim 1, wherein the GLP-1 agonist ~~or related molecule~~ is administered as a depot formulation.

11. (Currently amended) The method of claim 1, wherein the GLP-1 agonist ~~or related molecule~~ is administered to the mammal as a bolus at least about once daily.

12. (Currently amended) The method of claim 11, wherein the GLP-1 agonist ~~or related molecule~~ is administered to the mammal as a bolus at least once a week.

13. (Currently amended) The method of claim 1, wherein the administration of the GLP-1 agonist ~~or related molecule~~ is about twice daily (i.v. or subQ) for between from about one to about twenty weeks.

14. (Currently amended) The method of claim 1, wherein the method further comprises administering to the mammal a second therapeutically effective amount of the GLP-1 agonist ~~or a related molecule~~ following the drug holiday.

15. (Currently amended) The method of claim 14, wherein the method further comprises reducing administration of the second therapeutically effective amount of the

GLP-1 agonist ~~or related molecule~~ for a time conducive to producing a second drug holiday.

16. (Original) The method of claim 1 or 15, wherein the administration and reducing steps are repeated at least once.

17. (Original) The method of claim 16, wherein the administration and reducing steps are repeated at least about 2 to about 25 times.

18. (Original) The method of claim 17, wherein the administration and reducing steps are repeated as needed to prevent or treat the diabetes or related disorder.

19. (Original) The method of claim 18, wherein the method is practiced over the lifetime of the mammal.

20. (Currently amended) The method of claim 1, wherein the GLP-1 agonist ~~or related molecule~~ is administered to the mammal at a dose of at least about 0.01 nmol/kg (body weight).

21. (Currently amended) The method of claim 1, wherein the GLP-1 agonist ~~or related molecule~~ is selected from the group consisting of:

des Ser^{39}-exendin-4(1-39)-Lys_6-NH_2 (SEQ ID NO:25),

des Pro^{36}-exendin-4(1-39)-Lys_6-NH_2 (SEQ ID NO:5; COMPOUND 1),

des Ala^{35}-exendin-4(1-39)-Lys_6-NH_2 (SEQ ID NO:27),

des Gly^{34}-exendin-4(1-39)-Lys_6-NH_2 (SEQ ID NO:28),

des Ser^{39}-(Lys^{40}(palmitoyl))-exendin-4(1-39)-Lys_7-NH_2 (SEQ ID NO:29),

des Gly^{34}-(Lys^{40}(palmitoyl))-exendin-4(1-39)-Lys_7-NH_2 (SEQ ID NO:30),

des Ala^{35}-(Lys^{40}(palmitoyl))-exendin-4(1-39)-Lys_7-NH_2 (SEQ ID NO:31),

des Pro^{36}-(Lys^{40}(palmitoyl))-exendin-4(1-39)-Lys_7-NH_2 (SEQ ID NO:32),

Lys^{40}(palmitoyl)-exendin-4(1-39)-Lys_7-NH_2 (SEQ ID NO:33),

des Pro^{36},Pro^{37}-exendin-4(1-39)-Lys_6-NH_2 (SEQ ID NO:34),

Lys_6-des Pro^{36},Pro^{37},Pro^{38}-exendin-4(1-39)-NH_2 (SEQ ID NO:35),

Asn-$(Glu)_5$-des Pro^{36},Pro^{37},Pro^{38}-exendin-4(1-39)-NH_2 (SEQ ID NO:36),

Lys_6-des Pro^{36},Pro^{37},Pro^{38}-exendin-4(1-39)-Lys_6-NH_2 (SEQ ID NO:37),

Asn-$(Glu)_5$-des Pro^{36},Pro^{37},Pro^{38}-exendin-4(1-39)-Lys_6-NH_2 (SEQ ID NO:39),

des Pro^{36},Pro^{37},Pro^{38}-exendin-4(1-39)-Lys_6-NH_2 (SEQ ID NO:6),

Gly^8-GLP-1(7-36)-Lys_6-NH_2 (SEQ ID NO:7),

Lys_6-Gly^8-GLP-1(7-36)-Lys_6-NH_2 (SEQ ID NO:8),

Lys_6-Gly^8-GLP-1(7-36)-NH_2 (SEQ ID NO:9),

(Gly^8,Lys^{37}(palmitoyl))-GLP-1(7-36)(human)-Lys_7-NH_2 (SEQ ID NO:10),

(Gly^8,Lys^{26}(palmitoyl))-GLP-1(7-36)(human)-Lys_6-NH_2 (SEQ ID NO:11),

Gly^8,Lys^{34}(palmitoyl)-GLP-1(7-36)(human)-Lys_6-NH_2 (SEQ ID NO:12),

Gly^8-GLP-1(7-36)-Lys_8-NH_2 (SEQ ID NO:13),

Gly^8-GLP-1(7-36)-Lys_{10}-NH_2 (SEQ ID NO:14), and

Gly^8-GLP-1(7-37)-Lys_6-NH_2 (SEQ ID NO:15),

or the free acid or pharmaceutically acceptable salt thereof.

22. (Currently amended) The method of claim 1, wherein the GLP-1 agonist ~~or related molecule~~ is exendin-4, exendin-3; or an analog or derivative thereof, wherein said analog or derivative comprises an amino acid sequence at least 90% identical to exendin-4 or a fragment thereof, and said analog, derivative, or fragment increases endogenous insulin production.

23. (Cancelled)

24. (Previously presented) The method of claim 1, wherein the method further comprises administering at least one anti-diabetic drug to the mammal.

25. (Original) The method of claim 24, wherein the administration is below about a therapeutically effective amount for at least one of the drugs in the mammal.

26. (Original) The method of claim 24, wherein the administration is at least about at a therapeutically effective amount for at least one of the drugs in the mammal.

27. (Previously presented) The method of claim 24, wherein administration of the anti-diabetic drug is before or after the drug holiday.

28. (Currently amended) The method of claim 24, wherein at least one of the anti-diabetic drugs is insulin, an insulin analog; or a pharmaceutically acceptable mixture thereof, wherein said insulin analog is a recognized anti-diabetic drug.

29. (Previously presented) The method of claim 28, wherein the insulin or insulin analog is human insulin or a human insulin analog, bovine insulin or a bovine insulin analog, porcine insulin or a porcine insulin analog; or a mixture thereof.

30. (Previously presented) The method of claim 29, wherein the insulin analog is Lys (B28), Pro (B29) human insulin.

31. (Withdrawn) The method of claim 1, wherein the anti-diabetic drug is a sulfonylurea, biguanide, thiazolidinedione, diazoxide, somatostatin, or an alpha-glucosidase inhibitor.

32. (Withdrawn) The method of claim 31, wherein the sulfonylurea is selected from the group consisting of tolbutamide, chlorpropamide, tolazamide, acetohexamide, glyburide, glipizide, and gliclazide.

33. (Withdrawn) The method of claim 31, wherein the biguanide is metformin or phenformin.

34. (Withdrawn) The method of claim 31, wherein the thiazolidinedione is ciglitazone or pioglitazone.

35. (Withdrawn) The method of claim 31, wherein the alpha-glucosidase inhibitor is acarbose.

36. (Previously presented) The method of claim 1, wherein the mammal is a human subject who has or is suspected of having diabetes mellitus or a related disorder.

37. (Previously presented) The method of claim 36, wherein the diabetes mellitus is selected from the group consisting of insulin-dependent diabetes mellitus (IDDM or type I diabetes) and non-insulin-dependent diabetes mellitus (NIDDM, or type II diabetes).

38. (Original) The method of claim 36, wherein the human subject suspected of having the diabetes mellitus is genetically pre-disposed to develop the disease.

39. (Currently amended) The method of claim 36, wherein the disorder related to diabetes mellitus is selected from the group consisting of impaired glucose tolerance (IGT), maturity-onset diabetes of youth (MODY); leprechaunism (insulin receptor mutation), tropical diabetes, diabetes secondary to a pancreatic disease or surgery;

~~diabetes associated with a genetic syndrome (eg.,~~ Prader-Willi syndrome ~~syndrome)~~; pancreatitis; and diabetes secondary to endocrinopathies; adipositas; and metabolic syndrome (syndrome X).

40-78. (Cancelled)